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* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Apr 08   "Ask CAS" for self-help around the clock
 NEWS  3  Apr 09   BEILSTEIN: Reload and Implementation of a New Subject Area
 NEWS  4  Apr 09   ZDB will be removed from STN
 NEWS  5  Apr 19   US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
 NEWS  6  Apr 22   Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
 NEWS  7  Apr 22   BIOSIS Gene Names now available in TOXCENTER
 NEWS  8  Apr 22   Federal Research in Progress (FEDRIP) now available
 NEWS  9  Jun 03   New e-mail delivery for search results now available
 NEWS 10  Jun 10   MEDLINE Reload
 NEWS 11  Jun 10   PCTFULL has been reloaded
 NEWS 12  Jul 02   FOREGE no longer contains STANDARDS file segment
 NEWS 13  Jul 22   USAN to be reloaded July 28, 2002;
                   saved answer sets no longer valid
 NEWS 14  Jul 29   Enhanced polymer searching in REGISTRY
 NEWS 15  Jul 30   NETFIRST to be removed from STN
 NEWS 16  Aug 08   CANCERLIT reload
 NEWS 17  Aug 08   PHARMAMarketLetter(PHARMAML) - new on STN
 NEWS 18  Aug 08   NTIS has been reloaded and enhanced
 NEWS 19  Aug 19   Aquatic Toxicity Information Retrieval (AQUIRE)
                   now available on STN
 NEWS 20  Aug 19   IFIPAT, IFICDB, and IFIUDB have been reloaded
 NEWS 21  Aug 19   The MEDLINE file segment of TOXCENTER has been reloaded
 NEWS 22  Aug 26   Sequence searching in REGISTRY enhanced
 NEWS 23  Sep 03   JAPIO has been reloaded and enhanced

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

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                                                       ENTRY    SESSION
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STRUCTURE FILE UPDATES:    6 SEP 2002  HIGHEST RN 447682-31-7
DICTIONARY FILE UPDATES:   6 SEP 2002  HIGHEST RN 447682-31-7
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TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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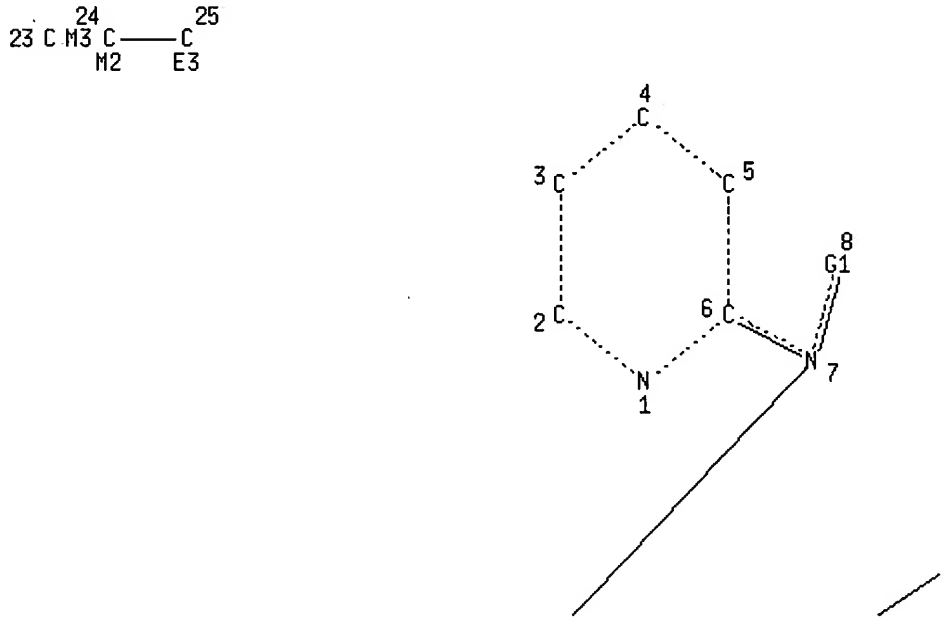

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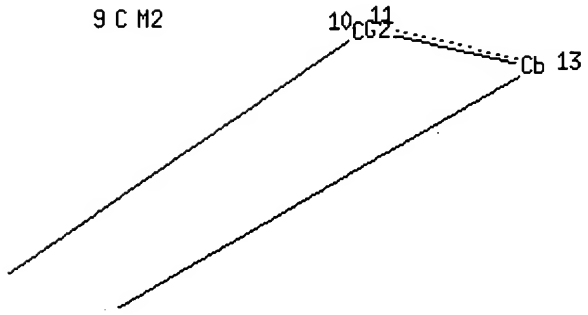

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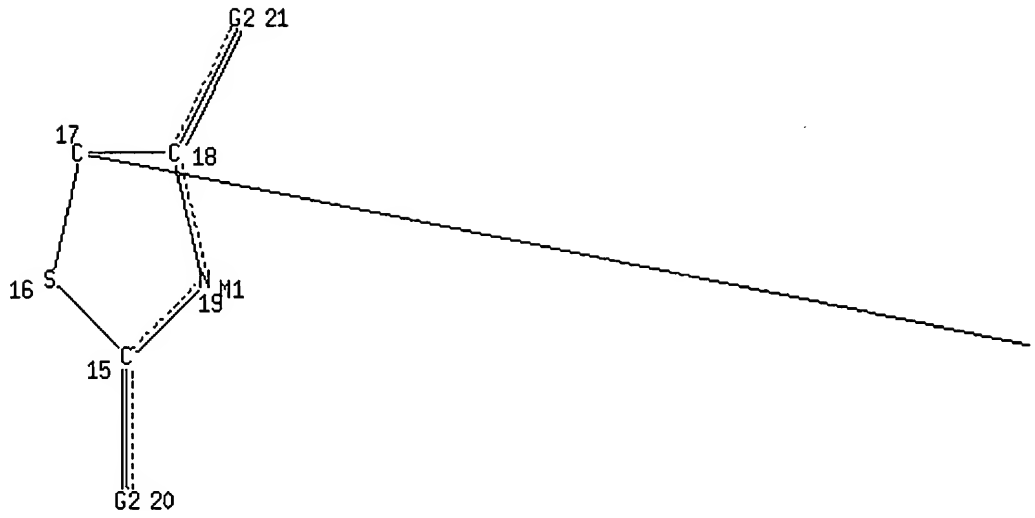

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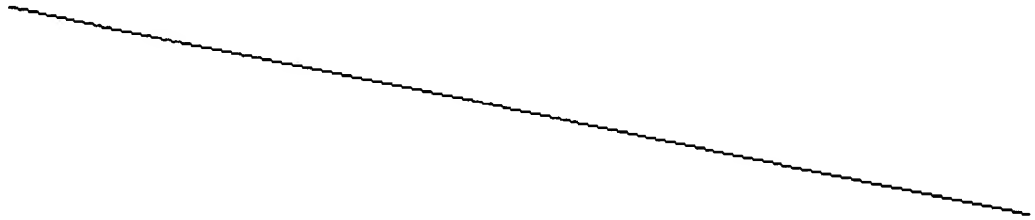

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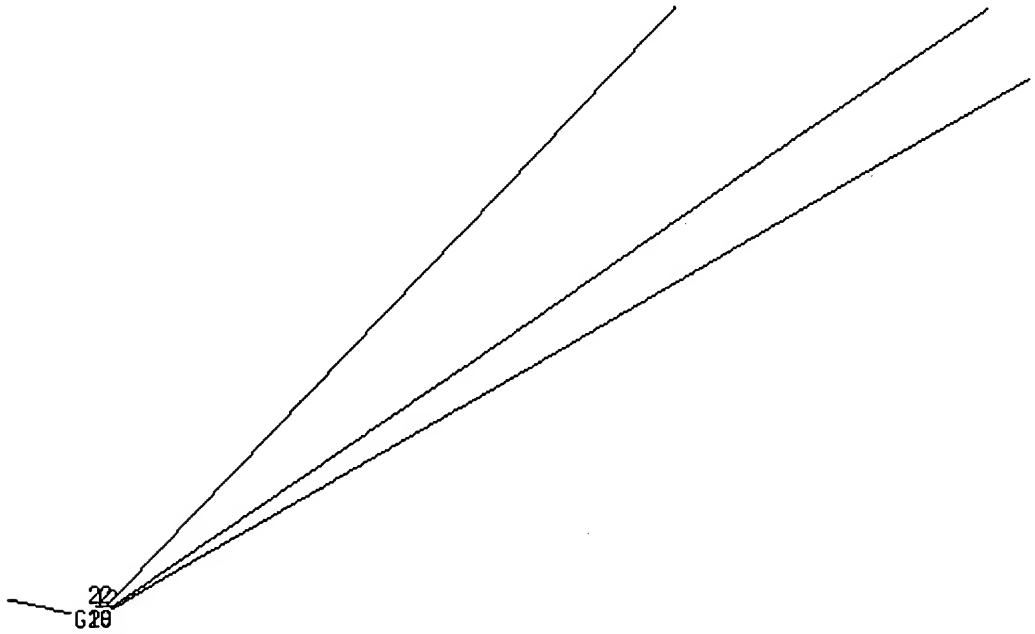

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GRAPH ATTRIBUTES:
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STEREO ATTRIBUTES: NONE


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FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
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PROJECTED ANSWERS:               2 TO      124

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FILE COVERS 1907 - 8 Sep 2002  VOL 137 ISS 11
FILE LAST UPDATED: 6 Sep 2002  (20020906/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

CAS roles have been modified effective December 16, 2001. Please
check your SDI profiles to see if they need to be revised. For
information on CAS roles, enter HELP ROLES at an arrow prompt or use
the CAS Roles thesaurus (/RL field) in this file.

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L8             3 L4 AND POLYMORPH

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L8   ANSWER 1 OF 3  HCAPLUS  COPYRIGHT 2002 ACS
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Full Text | Citing References

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ACCESSION NUMBER:         2000:772629  HCAPLUS
DOCUMENT NUMBER:          133:340315
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TITLE: Therapeutic action and properties of a polymorphic form of 5-[4-[2-(N-methyl-N-(2-pyridyl)amino)ethoxy]benzyl]thiazolidine-2,4-dione, maleic acid salt

INVENTOR(S): Blackler, Paul David James; Browne, Christine Marie; Coakley, Timothy G.; Giles, Robert Gordon; Morrissey, Gillian

PATENT ASSIGNEE(S): SmithKline Beecham PLC, UK; SmithKline Beecham (Cork) Limited

SOURCE: PCT Int. Appl., 21 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000064896	A1	20001102	WO 2000-GB1520	20000419
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1173435	A1	20020123	EP 2000-920892	20000419
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 2000009932	A	20020409	BR 2000-9932	20000419
NO 2001005147	A	20011217	NO 2001-5147	20011022

PRIORITY APPLN. INFO.:

GB 1999-9473	A	19990423
GB 1999-12196	A	19990525
WO 2000-GB1520	W	20000419

AB A polymorphic form of 5-[4-[2-(N-methyl-N-(2-pyridyl)amino)ethoxy]benzyl]thiazolidine-2, 4-dione, maleic acid salt (the **"Polymorph"**) characterized in that it provides: (i) an IR spectrum contg. peaks at 1763, 912, 856 and 709 cm^{-1}; and/or (ii) a Raman spectrum contg. peaks at 1762, 1284, 912 and 888 cm^{-1}; and/or (iii) a solid-state 13C NMR spectrum contg. peaks at 111.0, 113.6, 119.8, 129.1, 130.9, 131.8, 134.7, 138.7, 146.5, 152.7, 157.5, 169.5, 171.0, 178.7 ppm; and/or (iv) an x-ray powder diffraction (XRPD) pattern which gives calcd. lattice spacings at 5.87, 5.30, 4.69, 4.09, 3.88, 3.61, 3.53 and 3.46 Angstroms; a process for prepg. such a compd., a pharmaceutical compn. contg. such a compd. and the use of such a compd. in medicine.

IT **155141-29-0**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antidiabetic action and properties of polymorphic form of [[(N-methyl-N-(pyridyl)amino)ethoxy]benzyl]thiazolidinedione maleate)

RN 155141-29-0 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]-, (2Z)-2-butenedioate (1:1) (9CI) (CA INDEX NAME)

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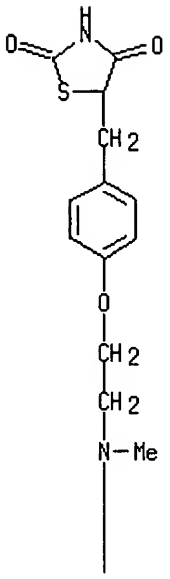
CRN 122320-73-4
CMF C18 H19 N3 O3 S

PAGE 1-A



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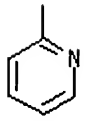

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CRN 110-16-7
CMF C4 H4 O4
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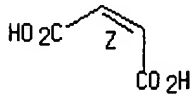

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 2000:772627 HCAPLUS
DOCUMENT NUMBER: 133:340314
TITLE: Therapeutic action and properties of a polymorphic form of 5-[4-[2-(N-methyl-N-(2-pyridyl)amino)ethoxy]benzyl]thiazolidine-2,4-dione, maleic acid salt
INVENTOR(S): Blackler, Paul David James; Giles, Robert Gordon; Moore, Stephen; Sasse, Michael John
PATENT ASSIGNEE(S): SmithKline Beecham PLC, UK
SOURCE: PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000064893	A2	20001102	WO 2000-GB1522	20000419
WO 2000064893	A3	20010125		
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RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1175418	A2	20020130	EP 2000-922793	20000419
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NO 2001005148	A	20011217	NO 2001-5148	20011022

PRIORITY APPLN. INFO.:		
GB 1999-9471	A	19990423
GB 1999-12195	A	19990525
WO 2000-GB1522	W	20000419

AB A polymorphic form of 5-[4-[2-(N-methyl-N-(2-pyridyl)amino)ethoxy]benzyl]thiazolidine-2,4-dione, maleic acid salt (the **"Polymorph"**) characterized in that it provides: (i) an infra red spectrum contg. peaks at 1752, 1546, 1154, 621, and 602 cm^{-1}; and/or (ii) a Raman spectrum contg. peaks at 1751, 1243 and 602 cm^{-1}; and/or (iii) a solid-state NMR spectrum contg. peaks at 111.9, 114.8, 119.6, 129.2, 134.0, 138.0, 144.7, 153.2, 157.1, 170.7, 172.0 and 175.0 ppm; and/or (iv) an x-ray powder diffraction (XRPD) pattern which gives calcd. lattice spacings of 6.46, 5.39, 4.83, 4.68, 3.71, 3.63, 3.58, and 3.48 Angstroms; a process for prepg. such a compd., a pharmaceutical compn. contg. such a compd. and the use of such a compd. in medicine.

IT **168553-12-6**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antidiabetic action of polymorphic form of [[(N-methyl-N-(pyridyl)amino)ethoxy]benzyl]thiazolidinedione maleate)

RN 168553-12-6 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]-, (2Z)-2-butenedioate (9CI) (CA INDEX NAME)

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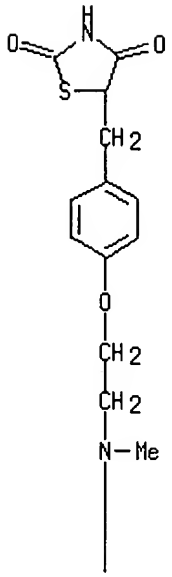
CRN 122320-73-4
CMF C18 H19 N3 O3 S

PAGE 1-A



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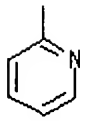

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CRN 110-16-7
CMF C4 H4 O4
CDES 2:Z

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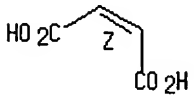

L8 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 2000:772626 HCAPLUS
DOCUMENT NUMBER: 133:340313
TITLE: Therapeutic action and properties of a polymorphic form of 5-[4-[2-(N-methyl-N-(2-pyridyl)amino)ethoxy]benzyl]thiazolidine-2,4-dione, maleic acid salt
INVENTOR(S): Blackler, Paul David James; Giles, Robert Gordon; Sasse, Michael John
PATENT ASSIGNEE(S): SmithKline Beecham P.L.C., UK
SOURCE: PCT Int. Appl., 18 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000064892	A2	20001102	WO 2000-GB1514	20000419
WO 2000064892	A3	20010125		
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RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
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NO 2001005149	A	20011217	NO 2001-5149	20011022

PRIORITY APPLN. INFO.:		
GB 1999-9472	A	19990423
GB 1999-12197	A	19990525
WO 2000-GB1514	W	20000419

AB A polymorphic form of 5-[4-[2-(N-methyl-N-(2-pyridyl)amino)ethoxy]benzyl]thiazolidine-2,4-dione, maleic acid salt (the "**Polymorph**") characterized in that it: (i) provides an IR spectrum contg. peaks at 1360, 1326, 1241, 714 and 669 cm^{-1}; and/or (ii) provides a Raman spectrum contg. peaks at 1581, 768, 670, 271 and 226 cm^{-1}; and/or (iii) provides a solid-state NMR spectrum contg. peaks at chem. shifts substantially; and/or (iv) provides an x-ray powder diffraction (XRPD) pattern contg. peaks; a process for prepg. such a compd., a pharmaceutical compn. contg. such a compd. and the use of such a compd. in medicine.

IT **168553-12-6**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antidiabetic action of polymorphic form of [[(N-methyl-N-(pyridyl)amino)ethoxy]benzyl]thiazolidinedione maleate)

RN 168553-12-6 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]-, (2Z)-2-butenedioate (9CI) (CA INDEX NAME)

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CRN 122320-73-4
CMF C18 H19 N3 O3 S

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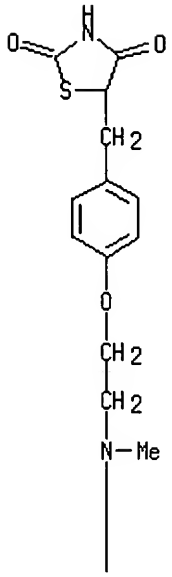

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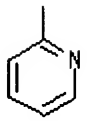

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CRN 110-16-7
CMF C4 H4 O4
CDES 2:Z

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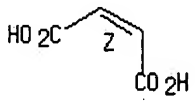

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L3 49 S L1 FULL

FILE 'HCAPLUS' ENTERED AT 19:42:19 ON 08 SEP 2002
L4 477 S L3
L5 153 S L4 AND PD < JUNE 1999
L6 0 S L5 AND BLACKER, P?/AU
L7 0 S L4 AND BLACKIER, P?/AU
L8 3 S L4 AND POLYMORPH

=> d l5, ibib abs fhitstr, 1-5

L5 ANSWER 1 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 2000:362595 HCAPLUS
DOCUMENT NUMBER: 133:13403
TITLE: Adipocyte containing ob gene promoter for screening modulators useful in treatment of anorexia, obesity, and other diseases
INVENTOR(S): Briggs, Michael R.; Auwerx, Johan; De Vos, Piet; Staels, Bart; Croston, Glenn E.; Miller, Stephen G.
PATENT ASSIGNEE(S): Ligand Pharmaceuticals Inc., USA
SOURCE: U.S., 64 pp., Cont.-in-part of U.S. Ser. No. 558,588, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6068976	A	20000530	US 1996-618100	19960319
CA 2215387	**AA**	**19960926**	**CA 1996-2215387**	**19960319**

PRIORITY APPLN. INFO.:

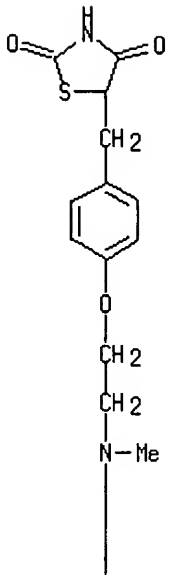
US 1995-408584	B2	19950320
US 1995-418096	B2	19950405
US 1995-510584	B2	19950802
US 1995-558588	B2	19951030
US 1995-7390P	P	19951121
US 1995-7721P	P	19951130
US 1995-8601P	P	19951214

AB This invention relates to the isolation and cloning of the promoter and other control regions of a human ob gene. It provides a method for identifying and screening for agents useful for the treatment of diseases and pathol. conditions affected by the level of expression of an ob gene. These agents interact directly or indirectly with the promoter or other control regions of the ob gene. A PPARγ agonist, BRL49653, has been identified to be useful in treating anorexia, cachexia, and other diseases characterized by insufficient food intake or body wt. loss. Modulators of ob gene expression may be used to treat other diseases such as obesity, diabetes, hypertension, cardiovascular diseases and infertility.

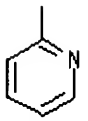
IT **122320-73-4**, BRL49653
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PPARγ agonist; adipocyte contg. ob gene promoter for screening modulators useful in treatment of anorexia, obesity, and other diseases)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 2-A



REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 2000:219115 HCAPLUS
DOCUMENT NUMBER: 132:231972
TITLE: Use of thiazolidinedione derivatives in the treatment of insulin resistance
INVENTOR(S): Antonucci, Tammy; Lockwood, Dean; Norris, Rebecca
PATENT ASSIGNEE(S): Warner-Lambert Company, USA
SOURCE: U.S., 15 pp., Cont.-in-part of U.S. Ser. No. 124,707.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6046202	A	20000404	US 1998-168515	19981008
US 5457109	**A**	**19951010**	**US 1994-292585**	**19940823**
US 5602133	**A**	**19970211**	**US 1995-469398**	**19950606**
US 6046222	A	20000404	US 1997-868608	19970604
US 5972944	A	19991026	US 1998-124707	19980729
AU 9952576	A1	19991202	AU 1999-52576	19991001

PRIORITY APPLN. INFO.:

US 1993-122251	B2	19930915
US 1994-292585	A3	19940823
US 1995-469398	A2	19950606
US 1996-763286	B2	19961210
US 1997-856987	A2	19970515
US 1997-868608	A3	19970604

US 1998-124707 A2 19980729
AU 1997-17709 A3 19970403

OTHER SOURCE(S): MARPAT 132:231972

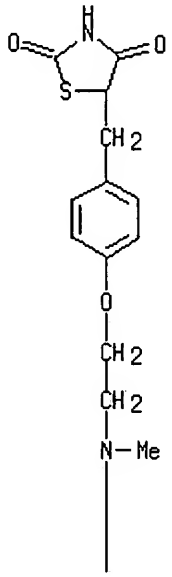
AB The invention provides methods of using a thiazolidinedione in the treatment of insulin resistance.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(thiazolidinedione derivs. for treatment of insulin resistance)

RN 122320-73-4 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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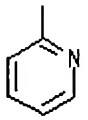


REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 2000:29169 HCAPLUS
DOCUMENT NUMBER: 132:288259
TITLE: Rosiglitazone has no clinically significant effect on nifedipine pharmacokinetics
AUTHOR(S): Harris, Robert Z.; Inglis, Anne Marie L.; Miller, Ann K.; Thompson, Kathleen A.; Finnerty, Dana; Patterson, Scott; Jorkasky, Diane K.; Freed, Martin I.
CORPORATE SOURCE: Drug Metabolism and Pharmacokinetics, SmithKline Beecham Pharmaceuticals, King of Prussia, PA, 19406, USA
SOURCE: Journal of Clinical Pharmacology (**1999**), 39(11), 1189-1194

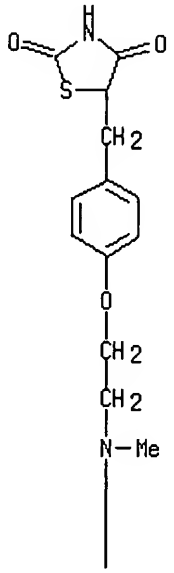
CODEN: JCPCBR; ISSN: 0091-2700
PUBLISHER: Sage Publications
DOCUMENT TYPE: Journal
LANGUAGE: English

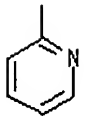
AB To examine the effects of repeat oral dosing of rosiglitazone on the pharmacokinetics of nifedipine, a prototype CYP3A4 substrate, a randomized, open-label, crossover study was performed with two treatment phases sepd. by a washout period of at least 14 days. Twenty-eight healthy male volunteers received either a single 20 mg oral nifedipine dose or rosiglitazone 8 mg orally once daily for 14 days with a single 20 mg oral nifedipine dose administered on day 14. Plasma nifedipine concns. were detd. over the 24-h period following administration of the nifedipine doses. Lack of effect was defined as the demonstration that the 90% CI was contained entirely within a sym. 30% range either side of unity on the loge-scale. Following rosiglitazone + nifedipine administration, the area under the nifedipine concn.-time curve from time zero to infinity (AUC(0-∞)) was 13% lower than that after administration of nifedipine alone. This difference in nifedipine AUC(0-∞) was not deemed to be clin. significant since the 90% CI was contained within the protocol-defined 30% range (point est. for ratio of geometric means 0.87; 90% CI: 0.79, 0.96). Rosiglitazone had no marked effect on nifedipine peak plasma concn. (point est.: 0.99; 90% CI: 0.73, 1.34) or time to peak concn. compared with nifedipine alone. Rosiglitazone coadministration produced a small decrease in the mean nifedipine half-life (point est.: -0.77; 90% CI: mean difference -1.29 h, -0.25 h). Both treatment regimens were well tolerated and assocd. with a favorable safety profile. Rosiglitazone, at the highest dose used in clin. studies, produced a small, clin. insignificant decrease in nifedipine exposure. The very small effect on nifedipine pharmacokinetics suggests that rosiglitazone is an extremely weak inducer of CYP3A4, a characteristic that distinguishes rosiglitazone from troglitazone.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(rosiglitazone effect on nifedipine pharmacokinetics)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A





REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 2000:10630 HCAPLUS
DOCUMENT NUMBER: 132:44986
TITLE: Combinations of glitazones, biguanides, and optional sulfonylureas for treatment of diabetes
INVENTOR(S): Whitcomb, Randall Wayne
PATENT ASSIGNEE(S): Warner-Lambert Company, USA
SOURCE: U.S., 22 pp., Cont.-in-part of U.S. 5,859,037. CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6011049	A	20000104	US 1998-189132	19981109
US 5859037	**A**	**19990112**	**US 1997-970057**	**19971113**
WO 2000027401	A1	20000518	WO 1999-US18140	19990811
W: AE, AL, AU, BA, BB, BG, BR, CA, CN, CR, CU, CZ, DM, EE, GD, GE, HR, HU, ID, IL, IN, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9953473	A1	20000529	AU 1999-53473	19990811
EP 1128834	A1	20010905	EP 1999-939130	19990811
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.:

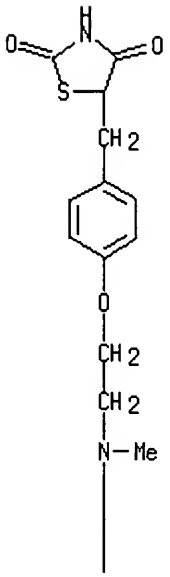
US 1997-38224P	P	19970219
US 1997-970057	A2	19971113
US 1998-189132	A	19981109
WO 1999-US18140	W	19990811

AB Combinations of a glitazone antidiabetic agent and a biguanide antidiabetic agent, and optionally a sulfonylurea antidiabetic agent, are useful for treating diabetes mellitus and improving glycemic control.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combinations of glitazones, biguanides, and optional sulfonylureas for diabetes treatment)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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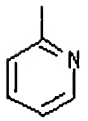


REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:810151 HCAPLUS
DOCUMENT NUMBER: 132:102683
TITLE: Therapeutic index for rosiglitazone in dietary obese rats: separation of efficacy and hemodilution
AUTHOR(S): Pickavance, L. C.; Tadayyon, M.; Widdowson, P. S.; Buckingham, R. E.; Wilding, J. P. H.
CORPORATE SOURCE: Department of Medicine, University of Liverpool, Liverpool, L69 3GA, UK
SOURCE: British Journal of Pharmacology (**1999**), 128(7), 1570-1576
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Stockton Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The blood glucose-lowering efficacy of rosiglitazone (RSG) and the mechanisms of assocd. wt. gain were detd. in dietary obese rats (DIOs). DIO and chow-fed rats received RSG 0.3-30 mg kg-1 daily for 21 days. In DIOs, plasma glucose and insulin concns. were reduced by RSG at dosages of 3 and 10 mg kg-1, resp. Homeostasis model assessment (HOMA) indicated the threshold for a redn. of insulin resistance was 1 mg kg-1. Neither glucose nor insulin levels were affected by treatment in chow-fed rats. RSG 0.3 mg kg-1 lowered free fatty acids (FFAs) in DIOs, whereas for plasma triglycerides (TGs), the threshold was 3 mg kg-1. By contrast, the threshold for reducing packed red cell vol. (PCV) and increasing cardiac mass was 10 mg kg-1. Thus, the therapeutic index for RSG in DIOs was >3 and ≤10. Energy intake and wt. gain increased in treated DIOs (by 20% and 50 g, at 30 mg kg-1) and chow-fed rats (by 25% and 35 g, at 30 mg

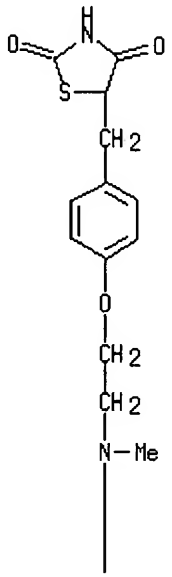
kg-1). In DIOs, these increases coincided with falls in plasma leptin (40% lower at 30 mg kg-1) and insulin (43% lower at 30 mg kg-1). By contrast, in chow-fed rats, wt. gain and hyperphagia occurred without changes in either leptin or insulin. However, redns. in FFAs below 0.4-0.3 mM were assocd. with hyperphagia and wt. gain in DIO and chow-fed rats. We conclude that increased energy intake and body wt. did not attenuate the improved metab. evoked by RSG in DIO rats, and that insulin action was enhanced at a dose >3 fold below the threshold for causing hemodilution and cardiac hypertrophy in DIO rats.

IT **122320-73-4**, Rosiglitazone
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(therapeutic index for rosiglitazone in dietary obese rats)

RN 122320-73-4 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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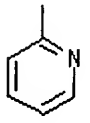

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L2 2 S L1
L3 49 S L1 FULL

FILE 'HCAPLUS' ENTERED AT 19:42:19 ON 08 SEP 2002

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L4          477 S L3
L5          153 S L4 AND PD < JUNE 1999
L6            0 S L5 AND BLACKER, P?/AU
L7            0 S L4 AND BLACKIER, P?/AU
L8            3 S L4 AND POLYMORPH

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L5 ANSWER 6 OF 153 HCAPLUS COPYRIGHT 2002 ACS

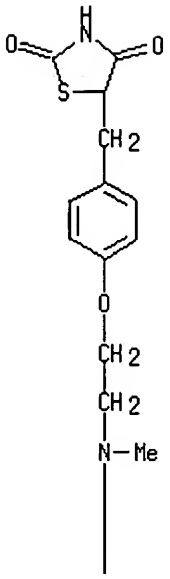
Full Text | Citing References

ACCESSION NUMBER: 1999:807214 HCAPLUS
DOCUMENT NUMBER: 132:146491
TITLE: Differential block by troglitazone and rosiglitazone of glibenclamide-sensitive K+ current in rat aorta myocytes
AUTHOR(S): Mishra, S. K.; Aaronson, P. I.
CORPORATE SOURCE: St Thomas's Campus, The Guy's, Department of Pharmacology, King's College and St Thomas' Hospitals' Medical and Dental School, London, UK
SOURCE: European Journal of Pharmacology (**1999**), 386(1), 121-125
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Thiazolidinediones are insulin-sensitizing agents effective in controlling type II diabetes. These compds. also cause vasodilation. We evaluated the effects of the thiazolidinediones troglitazone and rosiglitazone on the glibenclamide-sensitive K+ current in freshly isolated rat aorta myocytes. Troglitazone inhibited this current in a concn.-dependent manner (IC50~1 μM). Rosiglitazone had a similar, but much less potent (IC50~20 μM) action. Block of the glibenclamide-sensitive K+ channels, in particular by troglitazone, may potentially affect the response of arteries to hypoxia and to certain endogenous and exogenous vasodilators.

IT **122320-73-4**, Rosiglitazone
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(differential block by troglitazone and rosiglitazone of glibenclamide-sensitive K+ current in aorta myocytes)
RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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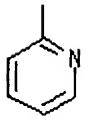


REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:799229 HCAPLUS
DOCUMENT NUMBER: 132:88016
TITLE: Studies on the euglycemic and hypolipidemic potentials of the novel indole analogue of thiazolidinedione, DRF 2189
AUTHOR(S): Chakrabarti, Ranjan; Vikramadithyan, Reeba Kannimel; Dileepkumar, Tripuraneni; Kumar, Kochunarayanapillai Bhadramma Sunil; Kumar, Mamnoor Prem; Misra, Parimal; Rao, Paraselli Bheema; Lohray, Vidya Bhusan; Lohray, Braj Bhusan; Rajagopalan, Ramanujam
CORPORATE SOURCE: Department of Pharmacology, Dr. Reddy's Research Foundation, Hyderabad, India
SOURCE: Arzneimittel-Forschung (**1999**), 49(11), 905-911
CODEN: ARZNAD; ISSN: 0004-4172
PUBLISHER: Editio Cantor Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Euglycemic and hypolipidemic activities of a novel indole analog of thiazolidinedione, DRF 2189 (CAS 172647-53-9), have been evaluated in different animal models. Compared to troglitazone (CAS 97322-87-7), DRF 2189 exhibited interesting plasma glucose and triglyceride lowering activity in genetically diabetic and obese db/db mice. It also produced a significant redn. in plasma glucose, triglyceride, total cholesterol levels and improvement in oral glucose tolerance in another genetic mouse model, the ob/ob mice. In high-fat diet fed Sprague-Dawley rats, DRF 2189 treatment showed improvement in plasma lipid parameters. Like other thiazolidinediones, this compd. also possesses peroxisome proliferator

activated receptor gamma (PPARγ) transactivation potential. In anesthetized rat expt., DRF 2189 produced a transient fall in blood pressure without any change in the ECG pattern. It showed non-specific smooth muscle relaxant activity against acetylcholine-, histamine- and potassium chloride-induced contractions in isolated guinea pig ileum. A twenty-eight-day toxicity study in Wistar rats did not show any signs of treatment-related adverse effects. The overall antidiabetic and hypolipidemic activities of DRF 2189 are comparable with rosiglitazone (CAS **155141-29-0**) and superior to troglitazone. In conclusion, results from these preclin. studies indicate that DRF 2189, a novel thiazolidinedione, has a marked potential for the management of type-2 diabetes.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

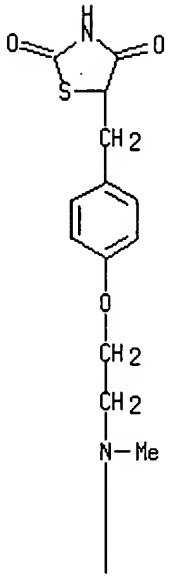
ACCESSION NUMBER: 1999:792188 HCAPLUS
DOCUMENT NUMBER: 132:18391
TITLE: Thiazolidinediones in the treatment of insulin resistance syndrome
AUTHOR(S): Cawthorne, M. A.
CORPORATE SOURCE: Clore Laboratory, University of Buckingham, Buckingham, MK18 1EG, UK
SOURCE: Progress in Obesity Research (**1999**), 8, 517-524
CODEN: POBREJ; ISSN: 0962-7936
PUBLISHER: John Libbey & Co. Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 14 refs. This article discusses the insulin sensitizing actions of thiazolidinediones, their mechanism of action, and preclin. and clin. effects in diabetes treatment.

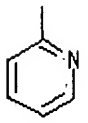
IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(thiazolidinediones in treatment of insulin resistance syndrome in humans)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 2-A



REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:789202 HCAPLUS
DOCUMENT NUMBER: 132:117393
TITLE: Chronic and acute effects of thiazolidinediones BM13.1258 and BM15.2054 on rat skeletal muscle glucose metabolism
AUTHOR(S): Furnsinn, C.; Brunmair, B.; Meyer, M.; Neschen, S.; Furtmuller, R.; Roden, M.; Kuhnle, H. F.; Nowotny, P.; Schneider, B.; Waldhausl, W.
CORPORATE SOURCE: Division of Endocrinology & Metabolism, Department of Medicine III, Vienna, A-1090, Austria
SOURCE: British Journal of Pharmacology (**1999**), 128(6), 1141-1148
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Stockton Press
DOCUMENT TYPE: Journal
LANGUAGE: English

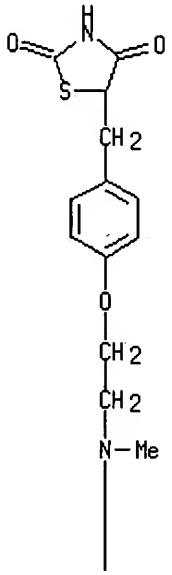
AB 1 New thiazolidinediones BM13.1258 and BM15.2054 were studied with regard to their PPARγ-agonistic activities and to their acute and chronic effects on glucose metab. in soleus muscle strips from lean and genetically obese rats. 2 Both BM13.1258 and BM15.2054 revealed to be potent PPARγ-activators in transient transfection assays in vitro. 3 In insulin-resistant obese rats, but not in lean rats, 10 days of oral treatment with either compd. increased the stimulatory effect of insulin on muscle glycogen synthesis to a similar extent (insulin-induced increment in μmol glucose incorporated into glycogen g-1 h-1: control, +1.19±0.28; BM13.1258, +2.50±0.20; BM15.2054, +2.55±0.46; $P<0.05$ vs control each). 4 In parallel to insulin sensitization, mean glucose

oxidn. increased insulin independently in response to BM13.1258 (to 191 and 183% of control in the absence and presence of insulin, resp.; P<0.01 each), which was hardly seen in response to BM15.2054 (to 137 and 124% of control, resp.; ns). 5 Comparable effects on PPARγ activation and on amelioration of insulin resistance by BM13.1258 and BM15.2054 were therefore opposed by different effects on glucose oxidn. 6 In contrast to chronic oral treatment, acute exposure of muscles to BM13.1258 or BM15.2054 in vitro elicited a distinct catabolic response of glucose metab. in specimens from both lean and obese rats. 7 The results provide evidence that BM13.1258 and BM15.2054 can affect muscle glucose metab. via more than one mechanism of action. 8 Further efforts are required to clarify, to what extent other mechanisms besides insulin sensitization via the activation of PPARγ are involved in the antidiabetic actions of thiazolidinediones.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(thiazolidinediones BM13.1258 and BM15.2054 chronic and acute effects on skeletal muscle glucose metab.)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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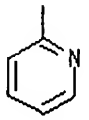


REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

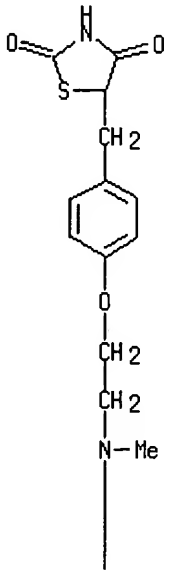
ACCESSION NUMBER: 1999:724649 HCAPLUS
DOCUMENT NUMBER: 132:202442

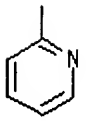
TITLE: Rosiglitazone: a new agent of the thiazolidinedione class for treatment of the type 2 diabetic patient
AUTHOR(S): Amato, Paul V.; Domenichini, David
CORPORATE SOURCE: Hartford Hospital, Hartford, CT, USA
SOURCE: Formulary (**1999**), 34(10), 825-826, 829-830, 832, 835
CODEN: FORMF9; ISSN: 1082-801X
PUBLISHER: Advanstar Communications, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 33 refs. Rosiglitazone is an orally active antidiabetic agent of the thiazolidinedione class. It was approved by the FDA in May, 1999, as monotherapy and in combination with metformin for the treatment of type 2 diabetic patients. As a potent agonist of peroxisome proliferator-activated receptor γ, rosiglitazone is theorized to improve glycemic control by improving insulin sensitivity in adipose tissue, skeletal muscle, and liver. Clin. trials of rosiglitazone as monotherapy and in combination with metformin, sulfonylureas, or insulin have shown clin. and significant effects on HbA1c and fasting blood glucose. The most common adverse effects have been respiratory tract infections, injury, and headache. Clin. data show no evidence of hepatotoxicity or elevations in liver enzymes. The usual starting dosage is 4 mg/day given once daily or in two divided doses; this dosage may be increased to 8 mg/day. Rosiglitazone appears to be an effective, safe, and competitively priced agent for the treatment of type 2 diabetics.

IT **122320-73-4**, Rosiglitazone
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(rosiglitazone: a new agent of the thiazolidinedione class for treatment of human type 2 diabetes)
RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A





REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

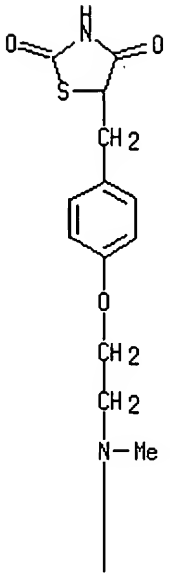
ACCESSION NUMBER: 1999:704526 HCAPLUS
DOCUMENT NUMBER: 132:59006
TITLE: Regulation of gene expression by activation of the peroxisome proliferator-activated receptor γ with rosiglitazone (BRL 49653) in human adipocytes
AUTHOR(S): Rieusset, Jennifer; Auwerx, Johan; Vidal, Hubert
CORPORATE SOURCE: Faculte de Medecine Rene Laennec, INSERM U449, Universite Claude Bernard Lyon-1, Lyon, 69372, Fr.
SOURCE: Biochemical and Biophysical Research Communications (**1999**), 265(1), 265-271
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To better define the mechanism of action of the thiazolidinediones, we incubated freshly isolated human adipocytes with rosiglitazone and investigated the changes in mRNA expression of genes encoding key proteins of adipose tissue functions. Rosiglitazone (10-6 M, 4 h) increased p85αphosphatidylinositol 3-kinase (p85αPI-3K) and uncoupling protein-2 mRNA levels and decreased leptin expression. The mRNA levels of insulin receptor, IRS-1, Glut 4, lipoprotein lipase, hormone-sensitive lipase, acylation-stimulating protein, fatty acid transport protein-1, angiotensinogen, plasminogen activator inhibitor-1, and PPARγ1 and γ2 were not modified by rosiglitazone treatment. Activation of RXR, the partner of PPARγ, in the presence of rosiglitazone, increased further p85αPI-3K and UCP2 mRNA levels and produced a significant augmentation of Glut 4 expression. Because p85αPI-3K is a major component of insulin action, the induction of its expression might explain, at least in part, the insulin-sensitizing effect of the thiazolidinediones. (c) 1999 Academic Press.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(gene expression of proteins involved in adipose tissue metab. as mechanism of PPAR γ-selective antidiabetic rosiglitazone in human adipocytes)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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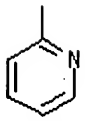

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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FILE 'REGISTRY' ENTERED AT 19:34:58 ON 08 SEP 2002
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L2 2 S L1
L3 49 S L1 FULL

FILE 'HCAPLUS' ENTERED AT 19:42:19 ON 08 SEP 2002
L4 477 S L3
L5 153 S L4 AND PD < JUNE 1999
L6 0 S L5 AND BLACKER, P?/AU
L7 0 S L4 AND BLACKIER, P?/AU
L8 3 S L4 AND POLYMORPH

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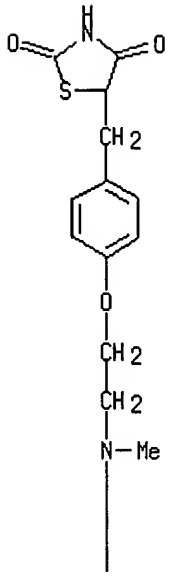
L5 ANSWER 20 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:617848 HCAPLUS
DOCUMENT NUMBER: 132:117298
TITLE: Peroxisome proliferator-activated receptor activators target human endothelial cells to inhibit leukocyte-endothelial cell interaction
AUTHOR(S): Jackson, Simon M.; Parhami, Farhad; Xi, Xiao-Ping; Berliner, Judith A.; Hsueh, Willa A.; Law, Ronald E.; Demer, Linda L.

CORPORATE SOURCE: Department of Medicine, University of California, Los Angeles, School of Medicine, Los Angeles, CA, 90095-1679, USA
SOURCE: Arteriosclerosis, Thrombosis, and Vascular Biology (**1999**), 19(9), 2094-2104
CODEN: ATVBFA; ISSN: 1079-5642
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An early event in acute and chronic inflammation and assocd. diseases such as atherosclerosis and rheumatoid arthritis is the induced expression of specific adhesion mols. on the surface of endothelial cells (ECs), which subsequently bind leukocytes. Peroxisome proliferator-activated receptors (PPARs), members of the nuclear receptor superfamily of transcription factors, are activated by fatty acid metabolites, peroxisome proliferators, and thiazolidinediones and are now recognized as important mediators in the inflammatory response. Whether PPAR activators influence the inflammatory responses of ECs is unknown. The authors show that the PPAR activators 15-deoxy-Δ12,14-prostaglandin J2 (15d-PGJ2), Wyeth 14643, ciglitazone, and troglitazone, but not BRL 49653, partially inhibit the induced expression of vascular cell adhesion mol.-1 (VCAM-1), as measured by ELISA, and monocyte binding to human aortic endothelial cells (HAECs) activated by phorbol 12-myristate 13-acetate (PMA) or lipopolysaccharide. The "natural" PPAR activator 15d-PGJ2 had the greatest potency and was the only tested mol. capable of partially inhibiting the induced expression of E-selectin and neutrophil-like HL60 cell binding to PMA-activated HAECs. Intracellular adhesion mol.-1 induction by PMA was unaffected by any of the mols. tested. Both PPAR-α and PPAR-γ mRNAs were detected in HAECs by using reverse transcription-polymerase chain reaction and a RNase protection assay; however, the authors have yet to det. which, if any, of the PPARs are mediating this process. Certain PPAR activators may thus help limit chronic inflammation mediated by VCAM-1 and monocytes without affecting acute inflammation mediated by E-selectin and neutrophil binding.
IT **122320-73-4**, BRL 49653
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(peroxisome proliferator-activated receptor activators target human endothelial cells to inhibit leukocyte-endothelial cell interaction)
RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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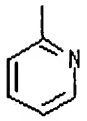


REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 21 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:607181 HCAPLUS
DOCUMENT NUMBER: 131:237812
TITLE: Cell culture conditions determine apolipoprotein CIII secretion and regulation by fibrates in human hepatoma HepG2 cells
AUTHOR(S): Clavey, Veronique; Copin, C.; Mariotte, M. C.; Bauge, E.; Chinetti, G.; Fruchart, J.; Fruchart, J. C.; Dallongeville, J.; Staels, B.
CORPORATE SOURCE: Faculte Pharmacie, Univ. Lille 2, Lille, Fr.
SOURCE: Cellular Physiology and Biochemistry (**1999**), 9(3), 139-149
CODEN: CEPBEW; ISSN: 1015-8987
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Fibrates are widely used drugs which lower triglycerides and increase HDL concns. in blood serum. Recent findings from our lab. have shown that fibrates repress apolipoprotein (apo) CIII gene expression, an effect that explains partially the triglyceride-lowering activity of these drugs. The effect of various fibrates on apo CIII gene expression in the human hepatoblastoma cell line HepG2 was studied. The authors demonstrate that the level of apo CIII secretion by HepG2 cells is controlled by serum factors whereas apo CIII mRNA levels are not and even increase under conditions when apo CIII secretion dramatically decreases. 12 Fetal calf serum batches were tested and apo CIII secretion in cell medium was only detected with 3 of them. The effect of serum on apolipoprotein secretion was more pronounced for apo CIII whereas other apolipoproteins (apo E, apo

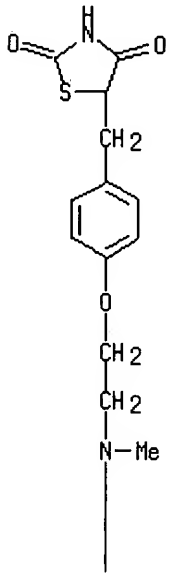
B, apo AII and apo AI) were affected to a lesser extent. Under serum conditions allowing apo CIII secretion, treatment with the peroxisome-proliferator activated receptor (PPAR)α activators fenofibrate, gemfibrozil and Wy-14643 result in a marked lowering of apo CIII secretion and gene expression, this effect being most pronounced with Wy-14643. Comparison of the activity of a PPARγ-specific ligand, the antidiabetic thiazolidinedione, BRL-49653 and a PPARα ligand Wy-14643 showed a marked decrease of apo CIII secretion and gene expression after activation of PPARA but not PPARγ. In conclusion, fibrates down-regulate apo CIII gene expression in human HepG2 cells, most likely via PPARα but not via PPARγ. However, these effects are only obsd. in HepG2 cells cultured under appropriate conditions.

IT **122320-73-4**, BRL-49653
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(antidiabetic effect on apolipoprotein secretion and regulation by fibrates in hepatoma HepG2 cells)

RN 122320-73-4 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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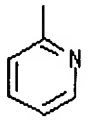


REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 22 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:590400 HCAPLUS
DOCUMENT NUMBER: 132:121278
TITLE: Use of a PPAR gamma-specific monoclonal antibody to

demonstrate thiazolidinediones induce PPAR gamma receptor expression in vitro

AUTHOR(S): Su, Jui-Lan; Winegar, Deborah A.; Wisely, G. Bruce; Sigel, Carlie S.; Hull-Ryde, Emily A.

CORPORATE SOURCE: Department of Molecular Sciences, Glaxo Wellcome Research and Development, Research Triangle Park, NC, 27709, USA

SOURCE: Hybridoma (**1999**), 18(3), 273-280
CODEN: HYBRDY; ISSN: 0272-457X

PUBLISHER: Mary Ann Liebert, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

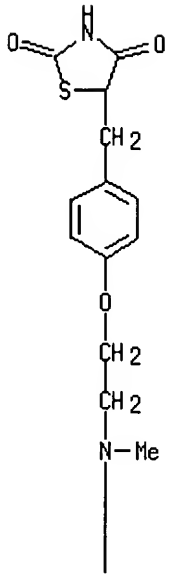
AB Troglitazone and rosiglitazone (BRL49653), members of the thiazolidinedione (TZD) class of antidiabetic drugs, are peroxisome proliferator-activated receptor γ (PPARγ) ligands that induce adipocyte differentiation and increase the expression of PPARγ protein. Here, we report the characterization of a PPARγ specific monoclonal antibody (MAb), PγA53.25, and its use to monitor PPARγ expression in the noncommitted pluripotent murine mesenchymal stem cell line, C3H10T1/2, treated with TZDs. MAb PγA53.25 was raised against a region in the N-terminal domain of human PPARγ shared by splice variants PPARγ1 and PPARγ2. It recognizes immunizing antigen in enzyme-linked immunoadsorbent assay (ELISA), and does not cross-react with the N-terminal domains of PPARα or PPARδ. In Western blotting, PγA53.25 reacts with the immunizing antigen as well as distinct protein bands corresponding to the mol. wt. of full length PPARγ from C3H10T1/2 cells and rat tissue lysates. In fluorescent microscopy, PγA53.25 immunostains nuclei of C3H10T1/2 cells treated with PPARγ ligands. The fluorescence intensity of the treated cells is TZD dose-dependent, and correlates with lipid accumulation consistent with adipogenesis. Based on these results, we propose that MAb PγA53.25 will be a useful tool for elucidating the role of PPARγ in fatty acid metab. and adipocyte differentiation.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of a PPAR gamma-specific monoclonal antibody to demonstrate thiazolidinediones induce PPAR gamma receptor expression in mesenchymal stem cells)

RN 122320-73-4 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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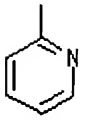

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L5 ANSWER 23 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:533775 HCAPLUS
DOCUMENT NUMBER: 131:266844
TITLE: A novel therapy for colitis utilizing PPAR-γ ligands to inhibit the epithelial inflammatory response
AUTHOR(S): Su, Chinyu G.; Wen, Xiaoming; Bailey, Shannon T.; Jiang, Wen; Rangwala, Shamina M.; Keilbaugh, Sue A.; Flanigan, Anne; Murthy, Sreekant; Lazar, Mitchell A.; Wu, Gary D.
CORPORATE SOURCE: Division of Gastroenterology, University of Pennsylvania School of Medicine, Philadelphia, PA, 19104, USA
SOURCE: Journal of Clinical Investigation (**1999**), 104(4), 383-389
CODEN: JCINAO; ISSN: 0021-9738
PUBLISHER: American Society for Clinical Investigation
DOCUMENT TYPE: Journal
LANGUAGE: English

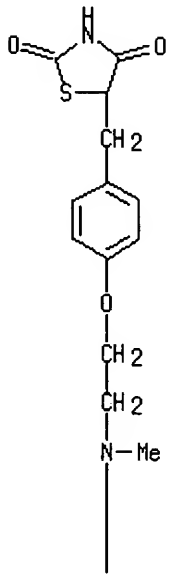
AB Peroxisome proliferator-activated receptor γ (PPAR-γ), a member of the nuclear hormone receptor super-family originally shown to play a crit. role in adipocyte differentiation and glucose homeostasis, has recently been implicated as a regulator of cellular proliferation and inflammatory responses. Colonic epithelial cells, which express high levels of PPAR-γ protein, have the ability to produce inflammatory cytokines that may play a role in inflammatory bowel disease (IBD). We report here that PPAR-γ ligands dramatically attenuate cytokine gene

expression in colon cancer cell lines by inhibiting the activation of nuclear factor-κB via an IκB-α-dependent mechanism. Moreover, thiazolidinedione ligands for PPAR-γ markedly reduce colonic inflammation in a mouse model of IBD. These results suggest that colonic PPAR-γ may be a therapeutic target in humans suffering from IBD.

IT **122320-73-4**, BRL 49653
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(a novel therapy for colitis utilizing PPAR-γ ligands to inhibit the epithelial inflammatory response)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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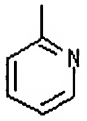

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L5 ANSWER 24 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:467127 HCAPLUS
DOCUMENT NUMBER: 131:237802
TITLE: Novel euglycemic and hypolipidemic agents: pyridine containing unsaturated thiazolidinediones
AUTHOR(S): Lohray, B. B.; Bhushan, Vidya; Reddy, A. Sekar; Rao, P. Bheema; Reddy, N. Jaipal; Reddy, K. Anantha; Vikramadithyan, Reeba K.; Rajagopalan, R.
CORPORATE SOURCE: Department of Medicinal Chemistry and Drug Discovery, Dr. Reddy's Research Foundation, Hyderabad, 500 050,

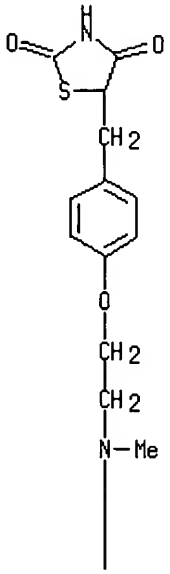
India
SOURCE: Indian Journal of Chemistry, Section B: Organic Chemistry Including Medicinal Chemistry (**1999**), 38B(4), 403-406
CODEN: IJSBDB; ISSN: 0376-4699
PUBLISHER: National Institute of Science Communication, CSIR
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Pyridyl contg. 2,4-thiazolidinediones having cyclic amine as linker have been synthesized. Both unsatd. thiazolidinedione 6 and satd. thiazolidinedione 5 and their various salts have been evaluated in db/db mice for euglycemic and hypolipidemic effects. The maleate salt of TZD 6a is found to be a very potent euglycemic and hypolipidemic compd.

IT **122320-73-4P**, BRL 49653
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(novel euglycemic and hypolipidemic agents: pyridine contg. unsatd. thiazolidinediones)

RN 122320-73-4 HCAPLUS
CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



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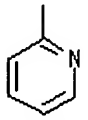


REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 25 OF 153 HCAPLUS COPYRIGHT 2002 ACS

Full Text | Citing References

ACCESSION NUMBER: 1999:446177 HCAPLUS
DOCUMENT NUMBER: 131:209060
TITLE: Rosiglitazone (BRL49653), a PPARγ-selective

agonist, causes peroxisome proliferator-like liver effects in obese mice

AUTHOR(S): Edvardsson, Ulrika; Bergstrom, Monica; Alexandersson, Maria; Bamberg, Krister; Ljung, Bengt; Dahllof, Bjorn

CORPORATE SOURCE: Cell Biology and Biochemistry, Astra Hassle AB, Moelndal, S-431 83, Swed.

SOURCE: Journal of Lipid Research (**1999**), 40(7), 1177-1184
CODEN: JLPRAW; ISSN: 0022-2275

PUBLISHER: Lipid Research, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

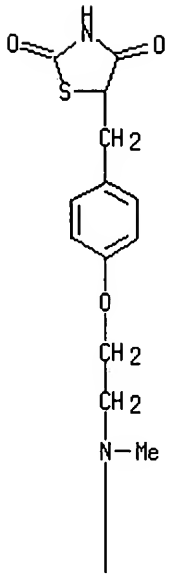
AB The PPAR (peroxisome proliferator activated receptor) transcription factors are ligand-activated nuclear receptors that regulate genes involved in lipid metab. and homeostasis. PPARα is preferentially expressed in liver and PPARγ preferentially in adipose tissue. Activation of PPARα leads to peroxisome proliferation and increased β-oxidn. of fatty acids in rodents. PPARγ-activation leads to adipocyte differentiation and improved insulin signaling of mature adipocytes. Both PPAR receptors are believed to be functional targets for treatment of hyperlipidemia in man. The authors have treated obese diabetic mice (ob/ob), which have highly elevated levels of plasma triglycerides, glucose and insulin, for 1 wk with WY14,643 (180 μmol/kg/day), a selective PPARα agonist, or rosiglitazone (BRL49653; 2.5 μmol/kg/day), a selective PPARγ agonist. The doses used produce a similar therapeutic effect in both treatment groups (lowering of triglycerides and glucose). High resoln. two-dimensional gel electrophoresis of livers showed that WY14,643 and rosiglitazone both produced changes in expression pattern of many proteins involved in peroxisomal fatty acid β-oxidn. However, similar expts. performed in lean mice showed significant up-regulation of these proteins only with WY14,643 treatment. Furthermore, the proteins up-regulated by the drugs in obese mice had a higher basal expression in obese controls compared to the lean littermates. Liver PPARγ mRNA levels were detd. and the authors obsd. that PPARγ2 mRNA levels were elevated in obese mice compared to lean littermates. As PPARα and PPARγ recognize similar DNA response elements, it is likely that the effects of rosiglitazone on PPARα responsive genes in livers of the ob/ob mice are mediated by PPARγ2.

IT **122320-73-4**, Rosiglitazone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PPARγ-selective agonist rosiglitazone (BRL49653) causes peroxisome proliferator-like liver effects in obese mice in relation to WY14,643 and hypolipemic and hypoglycemic effects)

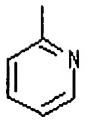
RN 122320-73-4 HCAPLUS

CN 2,4-Thiazolidinedione, 5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]- (9CI) (CA INDEX NAME)

PAGE 1-A



PAGE 2-A



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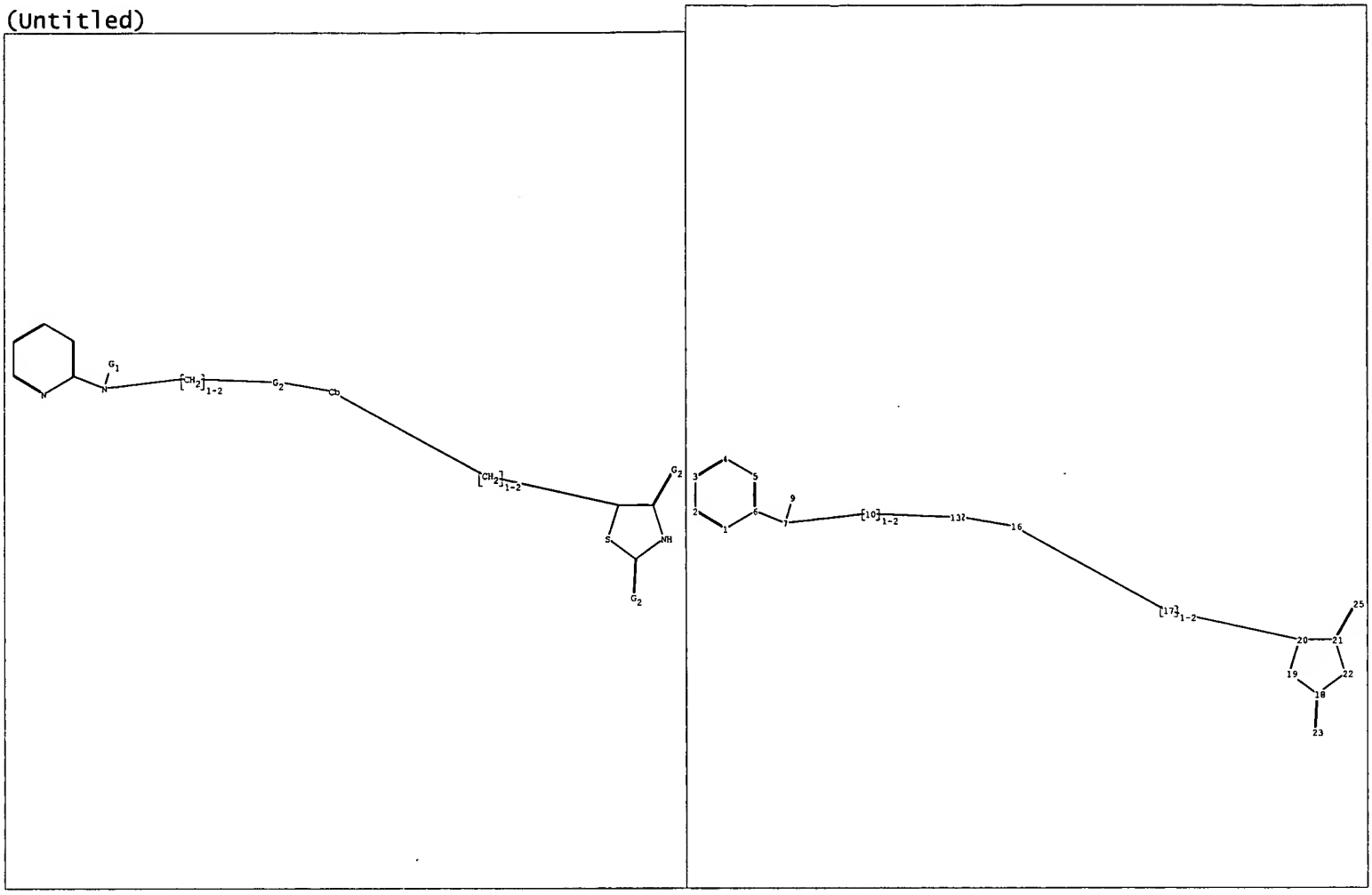

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exact bonds :
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isolated ring systems :
containing 1 : 18 :

G1:CH3,Et

G2:O,S

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C,C6